INCIDENCE AND RATE OF DISAPPEARANCE OF RETINAL HEMORRHAGE IN NEWBORNS

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Abstract.

INCIDENCE AND RATE OF DISAPPEARANCE OF RETINAL HEMORRHAGE IN NEWBORNS

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To determine the prevalence, associated biometric factors, and rate of disappearance of neonatal retinal hemorrhage, healthy newborns (n=149) at an urban hospital were examined using indirect ophthalmoscopy within 30 hours of birth. Newborns with retinal hemorrhage were re-examined biweekly until hemorrhage resolved. Intraretinal hemorrhage was present in 34% of newborns and varied from a single dot hemorrhage in one eye to bilateral widespread hemorrhages, occasionally with white centers. The incidence of hemorrhage was higher for vacuum-assisted (75%) than for spontaneous vaginal deliveries (33%), and was least for infants delivered by cesarean section (7%, $P<0.001$). The mean maternal age was greater for infants with retinal hemorrhage ($P<0.04$). By two weeks postpartum, retinal hemorrhage resolved in 86% of eyes, and at four weeks, no intraretinal hemorrhage was detected, though a single subretinal hemorrhage persisted until six weeks postpartum. Intraretinal hemorrhages are common in the immediate postnatal period and resolve by one month of age. Retinal hemorrhage in infants older than one month should heighten the suspicion that the hemorrhage is associated with factors other than birth.

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Introduction.

Since the initial detection in 1861 (1) of retinal hemorrhage (RH) in healthy newborns, presumably related to the birth process, several investigators have confirmed their presence. Reported incidences of neonatal RH vary widely between 2.6% and 50.5% (2-18). Previous investigations are also inconclusive regarding ophthalmoscopic features of neonatal RH and its relationship to neonatal and maternal biometric factors. Because RH in infants can be related to pathologic mechanisms other than the birth process, we sought to investigate the incidence, rate of disappearance, and associated biometric factors of neonatal RH.

I. Causal factors.

The variation in reported incidence and correlates of RH is rivaled by the number of attributed causes. Several theories to explain the occurrance of RH relate to pressure caused by uterine contractions and the trauma of delivery. Others focus on factors that predispose to trauma-induced damage or are not at all related to obstetrical trauma.

Direct obstetrical trauma.

Early investigators suggested that spastic uterine contractions and the narrow pelvic outlet canal caused increased intracranial pressure. These pressure elevations are transmitted along the intravaginal space surounding the optic nerve and retinal vein, and result in venous stasis, ophthalmic vein engorgement, capillary rupture, and hemorrhage (19). McKeown provides an anatomical explanation for the appearance of RH in the absence of conjunctival or choroidal hemorrhage, citing that although there may be anastomoses in a minority of cases, the central retinal vein usually drains into the cavernous sinus, which is most likely to experience cerebral compression during birth; in contrast, the conjunctival and choroidal venous systems anastomose with other drainage systems and are therefore not subject to the pressures and stasis of the cavernous sinus (14). The mechanism of cranial compression for RH has been supported by the finding of lower incidence of RH in preterm infants, because these neonates have smaller heads and therefore less compression (20). One study found increasing incidence of RH with larger head circumference (5). However, these roles for prematurity (5, 7, 8, 11) and head circumference (2, 21) have not been confirmed by others. In an attempt to address conclusively the issue, Svenningsen et al placed adjacent to the fetal head, an intrauterine pressure catheter, the readings of which are proportional to fetal

intracranial pressure. They found no relationship between incidence of RH and fetal head compression pressure duration or magnitude and concluded that the mechanism of RH is not elevated intracranial pressure (22).

Rather than emphasizing the magnitude of intracranial pressure, other authors have emphasized the *change* in intracranial pressure that occurs during labor and delivery as the cause of RH. In 1960, Giles proposed a mechanism of sudden release of intracranial pressure secondary to rapid delivery of the head over the perineum (10). This mechanism has been supported by correlations between RH and shorter length of stage II (active) labor and decreased birthweight, as evidence that smaller body size allows for rapid descent and faster pressure changes, resulting in RH (11, 16, 21). This relationship, however, between RH and length of labor (2, 23) and birthweight (2, 3, 5, 10, 12, 23, 24) has not been generally confirmed, and in some cases, a correlation between RH and increased birthweight is reported (7, 8). Levin et al focused on the correlation between RH and increased stage I of labor (cervical dilation), as evidence for long build-up of intracranial pressure followed by rapid release as the cause of RH (12). A correlation between RH and increased stage I of labor has been confirmed by others (3, 14).

Since the introduction of the Malmström vacuum extractor in 1954, vacuum-assisted deliveries have resulted in an increased incidence of RH in all studies in which the relationship has been adequately examined (3, 16, 18, 21, 25-27). Egge et al propose that the vacuum causes temporary venous stasis in the sagittal sinus and bridging veins, therefore increasing both the number and severity of hemorrhages (26). However, studies specifically examining the relationship between incidence of RH and duration of vacuum application have had mixed results (16, 24). In an effort to quantify indirectly intracranial strain, Svenningsen et al measured the strength and duration of traction forces associated with vacuum extraction, and found no relationship with incidence of RH (28). Based on these results, they suggest that the association between RH and vacuum extraction reflects a more intense fetal reaction to pain associated with vacuums, and pain results in increased venous pressure. Another study also concludes that RH in babies delivered by vacuum extraction is more a manifestation of a difficult labor and delivery rather than due to vacuum-induced trauma, based on the example of a case of RH in a neonate who delivered spontaneously right before the vacuum was applied (27).

Other factors that have been thought possibly to increase intracranial pressure include male gender and maternal primiparity, whereas administration of maternal analgesic may reduce the incidence by allowing for more maternal abdominal relaxation. However, the effect of gender (no effect (2, 5, 12, 24) or increased incidence in males (7, 14)), parity (no effect (2, 10, 12, 23, 24) or increased incidence in newborns of nulliparous mothers (3, 14, 16, 18)), and the use of analgesic (no effect (10, 12) or decreased incidence (23)) are all controversial.

The mode of delivery also has been though to play a role in the appearance of RH. Cesarean section is consistently associated with a reduced incidence of RH (3, 4, 8, 11, 12, 14, 16, 18, 27). Studies evaluating the use of forceps during delivery as a risk factor for retinal hemorrhages found either a greater risk (3, 14, 18), lesser risk (25, 26), or no effect (4, 10, 12, 24). Egge, et al proposed that forceps act like a helmet, thereby protecting the head and reducing the incidence of RH (26). Other studies suggest that the indication for forceps delivery inherently involves more stress and trauma than normal spontaneous vaginal deliveries, thereby resulting in increased rates of RH. The role of vacuum extraction in increasing the role of RH has already been discussed.

Edgerton attributes RH to direct obstetrical trauma, but not to intracranial pressure. He proposed that RH occurs after delivery of the head and is caused by venous congestion secondary to pressure of uterine contractions on the thorax (8). This mechanism, known as "traumatic asphyxia," is supported by other authors (24) and is thought to occur in adults following severe chest compression. However, traumatic asphyxia is associated with several other signs and symptoms, including a violaceous hue of the face and neck, diffuse edema of the face, and ocular findings including subconjunctival hemorrhage, macular edema, retinal hemorrhage, and vitreous hemorrhage, sometimes causing loss of vision (29). This distinct clinical picture has been reported only once in a newborn (30). The possibility that RH is caused by smaller degrees of chest compression that cause temporary cyanosis rather than traumatic asphyxia is contradicted by the absence of RH in five newborns with cyanosis (2).

Unrelated to obstetrical trauma.

Rather than attributing RH solely to trauma associated with labor and delivery, other authors have focused on factors that predispose to bleeding, such as vascular fragility, increased diapedesis due to

asphyxia, fetal anoxia, or prostaglandins, or bleeding diatheses. Other theories that relate RH to non-traumatic mechanisms include increased viscosity of blood, toxemia of pregnancy, and the first act of respiration; however, the proponents of these theories also recognize trauma as an independent cause of RH.

The importance of fragility of newborn retinal vasculature in the pathogenesis of RH has been thoroughly described (3). This is a possible explanation for the finding of an increased incidence of RH in preterm infants (8), because retinal angiogenesis occurs during the third trimester of pregnancy (31). To investigate the question further, Planten and Schaaf proposed that if retinal vessels were incompletely developed, then fragility would also affect other vascular beds, such as the urinary tract, thereby leading to presence of blood in urine (27). In 222 children, however, there was no correlation between urine blood and RH, leading the authors to conclude that incompletely developed vasculature is not the mechanism of RH.

Other authors have cited changes in normal vessels such as diapedesis due to asphyxia occurring during the transition from interruption of maternal circulation to the beginning of newborn circulation (3). Studies have used Apgar score and umbilical artery pH as signs of intrapartum asphyxia, a relationship that has been validated (32). The correlations between RH and fetal acidosis (21) and Apgar scores (3, 5, 33) have been confirmed by others. However, several studies have found no correlation between RH and umbilical artery pH (12, 23) or Apgar score (2, 12, 23, 24).

Fetal anoxia, resulting in fetal distress has been found to have positive (9), neutral (5) and negative (2, 4, 8, 10, 12, 16, 27) roles in relation to RH. Unfortunately, many of these studies may be confounded because fetal distress is often an indication for cesarean section, which independently results in a lower rate of RH (5).

On the basis of an increased incidence of RH following labor induced with dinoprostone (a PGE2 receptor agonist) rather than oxytocin, Schoenfeld et al proposed a different mechanism for diapedesis as the pathophysiology of RH in newborns (17). They proposed that prostaglandins, after transplacental transmission, cause RH by inducing vasodilation and increasing vascular permeability and intraocular pressure. The authors further speculate that the decreased incidence of RH in infants born by cesarean section is due to delivery before prostaglandin release takes place.

Several studies have focused on blood components rather than the vessels as the cause of RH. Lucas et al were the first to suggest that hypoprothrombinemia in neonates mediates RH (34). Because prothrombin concentration falls 2-9 days after birth (35), it was speculated that vitamin K administered to the mother during labor would be transmitted to the fetus and decrease the likelihood of RH. Initial studies showed a lower incidence of RH with the administration of vitamin K to mothers in labor (36). However, when the study was repeated in a blinded fashion, there was no effect on RH of vitamin K administration to the mother during labor and to the neonate following birth (9). Others have also found no relationship between the incidence of RH and coagulation deficit or clotting factor concentrations (2).

One study proposed that RH was due to increased blood viscosity of a high hemoatocrit condition found in neonates, similar to polycythemia vera in adults (2). The authors reach this conclusion without the foundation of an associated link between packed red cell volume and RH.

The relationship of toxemia of pregnancy to RH was first observed in 1958 (11), and the degree of toxemia has been noted to correlate with increased incidence of RH. In one study of thirty mothers with toxemia, the incidence of RH was 13.3% in newborns of mothers with pregnancy-induced hypertension or preeclampsia, and rose to 50% in newborns of mothers with eclampsia (37). This increase in incidence of RH with degree of toxemia has been confirmed by others (12); however, the finding that toxemia increases the incidence of RH is not universal (21).

The suggestion by Koenigstein in 1881 that RH occurs during the first act of respiration was not accompanied by a proposed mechanism, such as pressure changes secondary to the new pulmonary circulation, or altered blood chemistry due to the high oxygen content (38). More recent support for this mechanism has been quite limited.

In conclusion, numerous mechanisms have been suggested to account for the occurrence of neonatal RH; however, none of these are consistently supported by biometric data, and none explain all of the clinical characteristics of RH. Mechanical compression is a common, but not universal theme, as evidenced by the presence of RH in some newborns delivered by cesarean section. This most likely reflects a polyfactorial etiology of neonatal RH.

II. Type, location, and disappearance of hemorrhage.

The vast majority of reported neonatal RH is intraretinal and can be categorized in two common varieties: flame-shaped and dot-blot (2, 3, 10, 11, 14, 16, 18, 39). The more common type is flame-shaped, which is often found adjacent to the disc and along the vessels. This variety of hemorrhage is histologically described as superficial, lying between the axonal bundles within the nerve fiber layer of the retina. It is usually not found in the mid or peripheral retina due to the spreading of the nerve fiber layer bundles that form more of a reticular pattern beyond seven millimeters away from the optic disc (39). The second, less common type of RH noted in this population is dot-blot hemorrhage. This appears in the posterior pole or the periphery and lies deeper in the retina. Dot hemorrhage consists of a small cluster of red blood cells, whereas the darker, and usually bigger blot hemorrhage consists of full-thickness intraretinal hemorrhage (39). The locations, within the retina, of both flame-shaped and dot-blot hemorrhage have been confirmed by histological examination of two newborns who died with incidental findings of RH (14).

Less frequently, authors also report other types of RH in neonates. Some authors note rare, thin, sheetlike hemorrhages extending radially from the disc to the periphery, thought to be subhyaloid in location (10, 11, 14). One author describes lake or geographic hemorrhages, which are bright red and large, covering several disc areas (2); however, others believe that these are simply confluent dot-blot hemorrhages (21). The literature contains one report of a choroidal hemorrhage (27), and no reports of preretinal or vitreous hemorrhage.

Most authors agree that flame shaped hemorrhages are the predominant if not the only type found in mild cases of RH, whereas more severe cases have a mix of flame and dot-blot hemorrhage (11, 12, 21, 27). Fifty-four to 55% of cases of RH are bilateral (3, 5).

One study examined incidence of RH in separate cohorts of neonates at different times after birth; they found the incidence decreased from 18.9% within 24 hours to 12.5% from 25-48 hours, to 2.6% from 3-5 days after birth (18). Retinal hemorrhage in newborns of induced labors also resolve by 5 weeks of age (17). These findings suggest that the time-course of spontaneous clearance of neonatal RH may range from a few days to weeks. The rate of disappearance of RH, however, has not been systematically evaluated.

III. Pathologic significance.

Initially, substantial interest was given to neonatal RH in light of the unknown visual and neurologic sequelae. One author anecdotally reported that in 26 years of experience in following-up 2538 cases of neonatal RH, he never detected a case of amblyopia or other neurologic defect due to RH (11). Other reports which specified macular hemorrhage as a possible cause of amblyopia found no evidence of visual deficits in five cases of neonatal macular hemorrhage after four years (15). Possible amblyopia due to macular hemorrhage was found in one out of nine infants after ten years (40).

Reports on the neurologic and psychologic effects of RH have also shown no positive correlation. In a series of 317 newborns, Schenker found no correlation between RH and brain damage by neurological exam (16). Similarly, in 10 newborns with large RH, no evidence of intracranial hemorrhage was seen by computed tomography 2-4 days after birth (41). Longer-term studies of children who had or did not have RH found neither a developmental difference at 6-18 months (12), nor neurologic, psychologic, or ophthalmologic difference at 7 years of age (42).

IV. Relationship to child abuse

Although birth-related retinal hemorrhage does not by itself cause subsequent visual or neurological deficits, retinal hemorrhage in infants may be associated with systemic and other ocular pathology, including intracranial disorders, hematologic disturbances, chorioretinal infections, and retinal vascular diseases (39) (Figure 1). Intraretinal hemorrhages also occur in association with post-natal accidental or intentional trauma, and are a key feature of the shaken-baby syndrome (43-45) (Figure 1).

One study of 111 infants less than three years old with subdural hematomas who underwent ophthalmologic exams demonstrated intraretinal hemorrhage in 79% of cases (46). Preretinal (38%), subretinal (10%), and vitreous (14%) hemorrhage was also noted in these infants with subdural hematomas. Other findings included circular retinal folds and dome-shaped hemorhages under the internal limiting membrane. Prior episodes of abuse sometimes resulted in retinal scarring or hemosiderin deposition. Of the cases in which hemorrhages were observed, 87% were bilateral. Some of these features are not seen in neonatal RH.

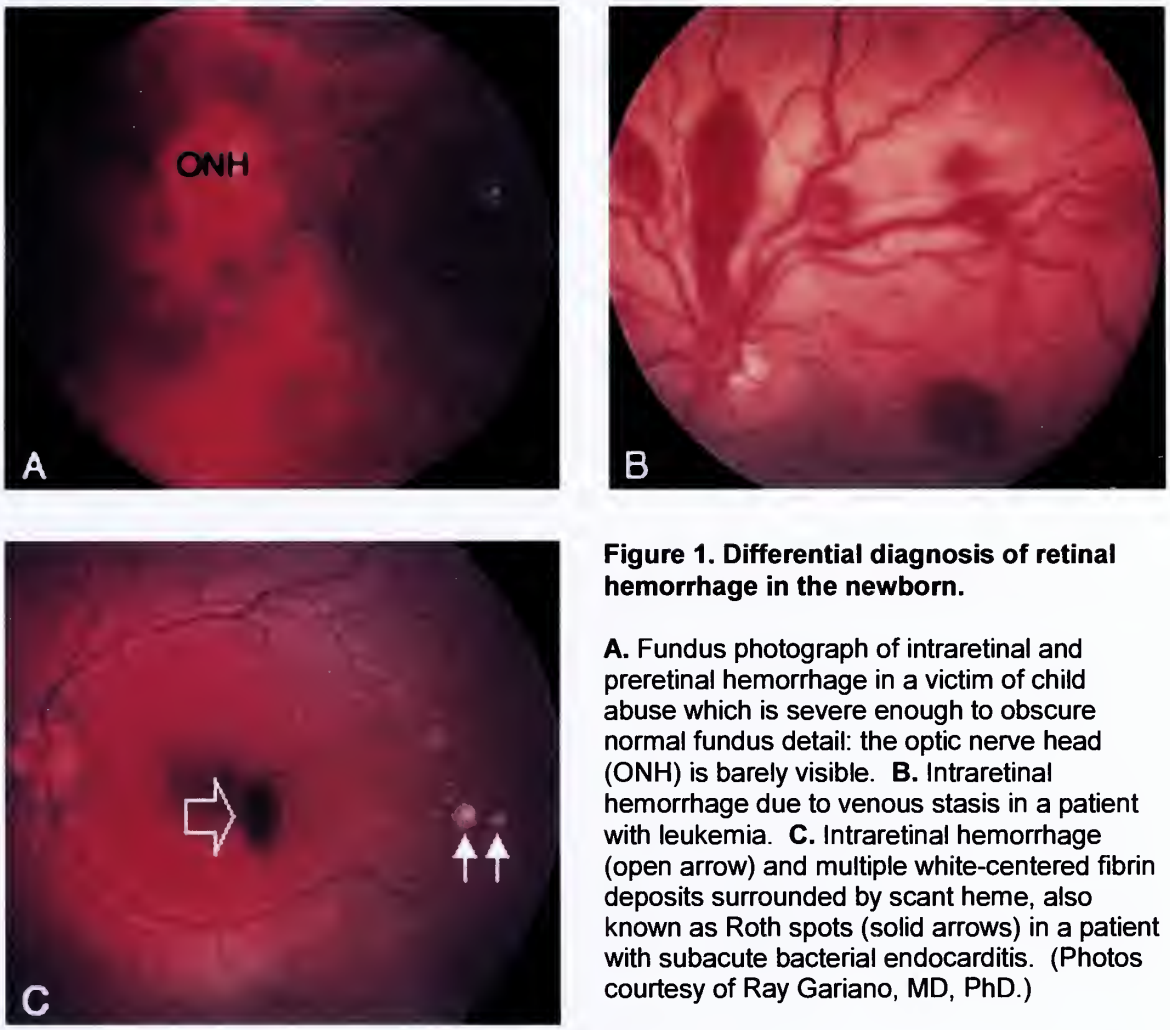

Figure 1. Differential diagnosis of retinal hemorrhage in the newborn.

A. Fundus photograph of intraretinal and preretinal hemorrhage in a victim of child abuse which is severe enough to obscure normal fundus detail: the optic nerve head (ONH) is barely visible. **B.** Intraretinal hemorrhage due to venous stasis in a patient with leukemia. **C.** Intraretinal hemorrhage (open arrow) and multiple white-centered fibrin deposits surrounded by scant heme, also known as Roth spots (solid arrows) in a patient with subacute bacterial endocarditis. (Photos courtesy of Ray Gariano, MD, PhD.)

Nonetheless, the ophthalmoscopic appearance of traumatic and birth related RH are often similar. Therefore, additional characteristics that distinguish between hemorrhages due to birth or to trauma may be critical in the assessment of possible child abuse. The time-course for spontaneous clearing of birth-related hemorrhage may facilitate differentiation of traumatic from birth-related RH. This information is missing from the literature.

Statement of Purpose and Hypothesis.

We hypothesize that birth-related retinal hemorrhage can be differentiated from retinal hemorrhage of child abuse based on ophthalmoscopic features and rate of disappearance.

Methods.

This study was conducted with institutional review board approval as Yale University School of Medicine Human Investigation Committee (HIC) Protocol Number 10299. Babies admitted to the newborn special care unit or with known or suspected systemic or ocular disease or congenital malformation were excluded. Subjects considered for enrollment were a cohort of 402 consecutive births over a period of 65 weekdays, admitted to the Well-Baby Nursery at the Children's Hospital of Yale-New Haven Medical Center, and determined by the pediatrics service to be free of any medical problems. One or both parents were verbally invited to participate in this study, and written informed consent was sought from one or both parents of each subject; permission was obtained also from the subject's pediatrician before enrollment in the study.

Enrolled newborns (n=149) were initially examined within 30 hours of birth, 30 minutes after topical instillation of phenylephrine 2.5% and tropicamide 0.5%. Examination was performed by a fellowship-trained retinal specialist using a pediatric eyelid speculum, binocular indirect ophthalmoscopy, and scleral depression with proparacaine 0.5% topical anesthetic. If no retinal hemorrhage was detected within 30 hours of birth, further evaluation was not scheduled. If hemorrhage was present, indirect ophthalmoscopy was repeated every two weeks until hemorrhage was no longer evident.

Hemorrhages were classified according to location in three retinal regions. Zone I encompasses one disc diameter around the optic nerve head and fovea, zone II extends from the anterior boundary of zone I to the equator, and, zone III is anterior to zone II, to the ora serrata (47).

Degree or severity of hemorrhage was designated by the number of hemorrhages per eye (48): Grade I = one or two hemorrhages; grade II = 3-10 hemorrhages; and grade III = greater than ten hemorrhages. The shapes, location, and other associated features of the hemorrhages were noted for each eye in a fundus drawing.

The following factors were assessed prior to the initial examination, and were unknown to the examiner:, maternal parity, episiotomy, analgesic, anesthetic, induction of labor, time since delivery, length of labor, mode of delivery, gestational age, birthweight, and head circumference.

Statistical analysis included use of two-tailed Student's t-tests for the comparison of continuous variables. Chi-square and ANOVA tests were used for comparisons with multiple variables. Correlation coefficients were calculated. Mean data are indicated ± standard deviation.

The topic of this study was proposed by Kathleen Stoessel, MD. The primary author (MVE) wrote the study protocol, obtained HIC approval, consented and enrolled all subjects, administered dilating and anesthetic drops, assisted in stabilizing each subject for fundus exam, scheduled and organized follow-up exams, collected maternal and neonatal biometric data, and performed all statistical analyses. Indirect ophthalmoscopy was performed by one of four retinal specialists (Ray Gariano, MD, PhD, Dante Pieramici, MD, Kathleen Stoessel, MD, or John Berreen, MD).

Results.

Of 402 consecutive newborns eligible for enrollment, parental consent was obtained for 149 (37.1%) infants, who were enrolled in this study, and underwent an initial retinal examination. Demographic features of the subject population are given in Table 1.

Table 1. Demographic features of the subject population.

Mode of delivery:	spontaneous vaginal	120 (80.5%)
	vacuum extraction	12 (8.1%)
	cesarean section	15 (10.1%)
	forceps assisted	2 (1.3%)
Mean maternal age (years)		27.8±6.25
Mean maternal parity		1.0±1.2
Mean length of stage II labor (min)		48.4±42.8
Mean birthweight (kg)		3.48±0.50
Mean gestational age (weeks)		37.8±1.2
Mean Apgar Score:	1 minute	8.6±0.8
	5 minute	9.0±0.2
Mean head circumference (cm)		34.4±1.9
Race:	white	86 (61.0%)
	black	36 (25.5%)
	hispanic	13 (9.2%)
	asian	6 (4.3%)
Male gender		76 (51%)
Episiotomy		39 (26.2%)
Induction of labor		85 (57.0%)
Maternal analgesic		99 (66.4%)

Retinal hemorrhage was found in 34% (50/149) of newborns. Of the 50 newborns with hemorrhage, 26 (52%) had hemorrhage in both eyes, so that 26% (76/298) of eyes examined contained hemorrhage. Hemorrhages were dot-blot or flame-shaped; larger blot hemorrhages often contained white centers (Figure 2). Hemorrhage was intraretinal in all subjects, except one, who had, in addition to intraretinal blood, a single zone I subretinal hemorrhage approximately 350 μm in diameter.

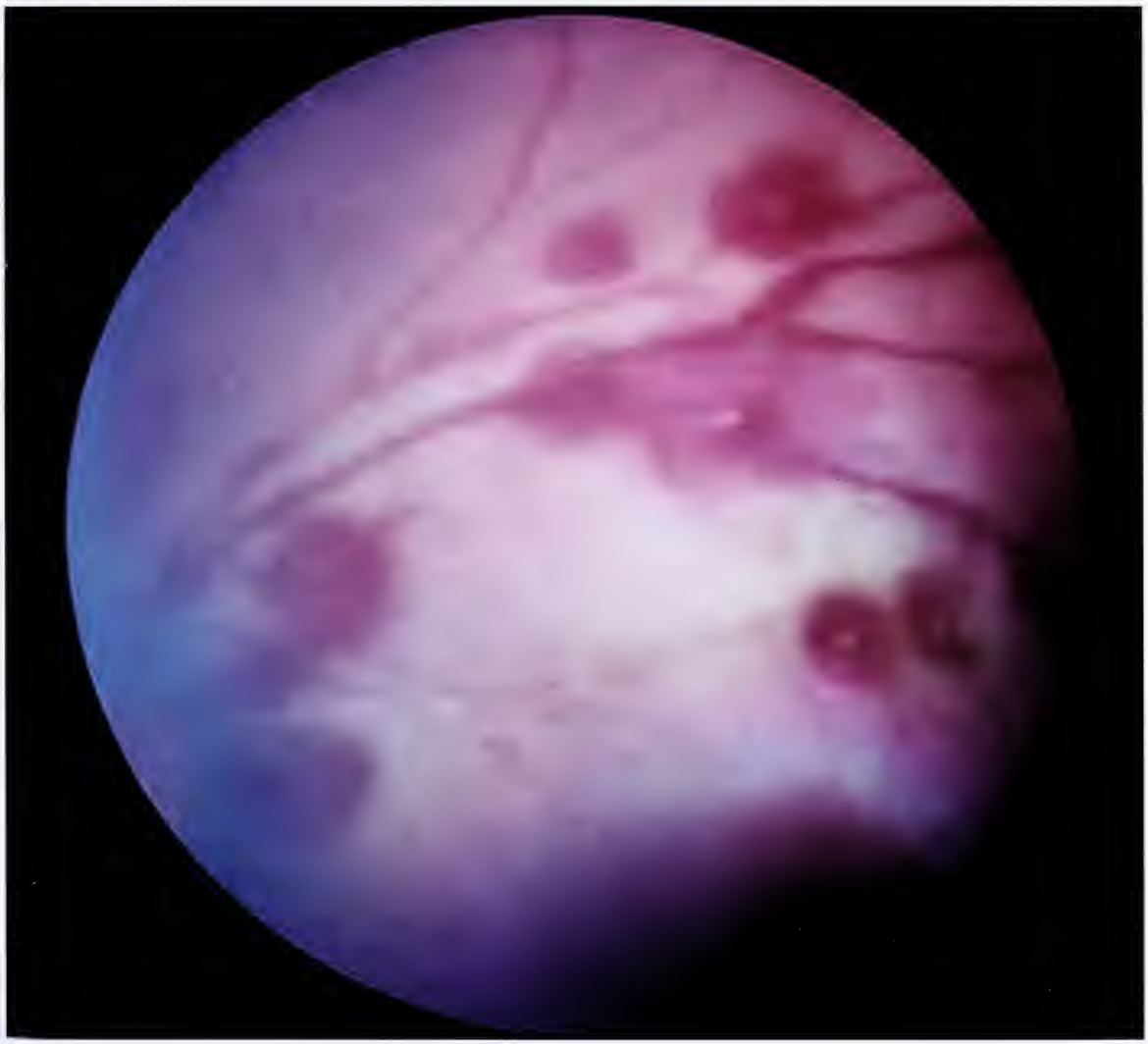

Figure 2. Fundus photograph of zones I and II, grade III, retinal hemorrhages in a baby girl 21 hours after birth. Note several of the larger hemorrhages contain white centers.

Of the 76 eyes with hemorrhage, 73 (96%) had hemorrhage in zone I, and 3 (4%) had hemorrhage in zone II only (n=2) or both zones II and III (n=1). Of the 73 eyes with hemorrhage in zone I, 19 (26%)

had hemorrhage in zone I only; 29 (40%) had hemorrhage in zones I and II only; 24 (33%) had hemorrhage in zones I, II, and III; and 1 (1.3%) had hemorrhage in zones I and III only.

Of the 24 newborns with one affected eye, hemorrhage appeared in zone I in 22 cases (92%). Hemorrhage was confined to zone I in 11 patients (46%), zones I and II in 6 patients (25%), and was present in all zones in 4 patients (17%). One newborn (4%) had hemorrhage in zone II only, and one (4%) in zones II and III only.

Of the 26 infants with bilateral hemorrhage, 14 (54%) exhibited the same zonal distribution of hemorrhage in both eyes: zone I only in 2 (8%), zones I and II in 6 (23%), or all zones in 6 (23%). Three infants (12%) had hemorrhage in zone I only in one eye and multiple zone involvement in the fellow eye. One newborn (4%) had hemorrhage in zone I and II in one eye, and zone II in the other eye, and 8 (31%) newborns had hemorrhage in zones I and II in one eye, and in all zones in the fellow eye.

The severity of hemorrhage was grade I in 17/76 (22%) eyes, grade II in 18/76 (24%) eyes, and grade III in 41/76 (54%) eyes. Of infants with hemorrhage in one eye only, 13 (54%) had grade I severity, 6(25%) had grade II severity, and 5(21%) had grade III severity. Of newborns with bilateral hemorrhage, 23 (88%) had grade III severity in at least one eye, and 17 (65%) bilaterally. The remaining infants had, in the two eyes, grades I and II (n=1), grades I and III (n=4), grades II and III (n=1), and grade II bilaterally (n=1). Severity of hemorrhage in one eye did not correlate with presence or severity of hemorrhage in the fellow eye ($r=+0.2$, $p=0.07$).

The incidence of hemorrhage was greatest for vacuum-assisted deliveries (9/12, or 75%), intermediate for spontaneous vaginal births (40/120, or 33%), and least for cesarean section deliveries (1/15, or 7%; $p<0.001$); Table 2). Two babies born by forceps-assisted delivery exhibited no retinal hemorrhage.

Mean maternal age was higher for infants with retinal hemorrhage than those without (29.1±5.7 years vs. 27.1±6.4 years; $p<0.04$, Student's t test). The incidence of retinal hemorrhage was not associated with race, parity, episiotomy, use of maternal analgesic or anesthetic, induction of labor, length of active labor, gestational age, Apgar scores, birthweight, head circumference, or gender (Table 2). There was no relationship between mode of delivery and maternal age by ANOVA ($p>0.3$).

Table 2. Incidence of retinal hemorrhage (RH) by maternal and neonatal biometric data.

		RH (+)	RH (-)
Mode of delivery:	spontaneous vaginal[A]	40 (33%)	80
	vacuum extraction[A]	9 (75%)	3
	Cesarean section[A]	1 (6.7%)	14
	forceps assisted	0 (0%)	2
Maternal age (years) [B]		29±5.7	27±6.4
Maternal parity		1.0±1.0	1.0±1.3
Length of stage II labor (min)		45±40	49±44
Birthweight (kg)		3.4±0.45	3.5±0.53
Gestational age (weeks)		37±1.1	37±1.2
Apgar Score:	1 minute	8.5±0.9	8.7±0.8
	5 minute	9.0±0.2	9.0±0.3
Head circumference (cm)		34±1.5	34±2.1
Race:	white	34(39%)	52
	black	10(27%)	26
	hispanic	3(23%)	10
	asian	2(33%)	4
Gender (male:female)		24:26	52:47
Episiotomy (yes:no)		13:37	26:73
Induction of labor (yes:no)		19:31	47:52
Maternal analgesic (yes:no)		14:36	36:63

RH = retinal hemorrhage

[A]$P<0.001$ by chi-square test

[B]$P<0.04$, by Student's t test

Twenty-six of 76 (34%) eyes (17 of 50 (34%) patients) with retinal hemorrhage were lost to follow-up. At two weeks after birth, retinal hemorrhages had disappeared entirely in 86% (43/50) of eyes, or 85% of patients (28/33). Intraretinal hemorrhages persisted in 14% (7/50) of eyes, and were markedly faded in all cases in comparison to their appearance on initial examination. All seven eyes with persistent hemorrhage two weeks after birth had grade III severity initially.

Five of the seven eyes (3 of 5 patients) with hemorrhage at two weeks were reexamined four weeks after birth. In four of these eyes, all hemorrhage disappeared within 4 weeks of birth. The only persistent hemorrhage was a single subretinal hemorrhage observed in the study; two weeks later, or six weeks after birth, this subretinal hemorrhage also was not detected.

Discussion.

The incidence of retinal hemorrhage in newborns in this study (34%) is comparable to several reported values (30% (4), 32.6% (9), 34% (17), 35% (3), 37.3% (12), 37.5% (5), 40% (10), 41.7% (8), and 50.5% (14)), but greater than values given in several studies (2.6% (6), 10.2% (13), 13% (7), 14.5% (15), 15% (2), 18.2% (11), 18.9% (18), and 19.2% (16)). Variation in reported values may be due to different patient demographics, variable time periods between birth and initial examination, inclusion of children with systemic diseases, and use of pupillary dilation, lid speculums, direct or indirect ophthalmoscopy, and scleral depression. To minimize the likelihood of RH being absorbed before initial exam, all initial exams were performed within 30 hours of birth. To allow for thorough exam, our protocol involved use of pupillary dilation, lid speculum, and indirect ophthalmoscopy with scleral depression.

In the majority of eyes, hemorrhages were located in the posterior pole (zone I), though they were also often found in more anterior regions. In a small number of newborns, hemorrhage was evident only anterior to zone I, and hemorrhage in this zone is unlikely to be detected without indirect ophthalmoscopy. Though in some infants isolated dot or flame-shaped hemorrhage was seen, most eyes exhibited greater than ten hemorrhages, and in some cases, hemorrhages were confluent or too numerous to count. Like the findings of others (3, 5), the severity of hemorrhage in one eye, however, was not predictive of presence or severity of hemorrhage in the fellow eye. White-centered hemorrhage was commonly noted (18/76 eyes, 24%).

Factors associated with a greater risk of retinal hemorrhage in this study were vacuum-assisted delivery and increased maternal age; the effect of maternal age, however, was small and of doubtful clinical meaning. Delivery by cesarean section appeared protective. Other maternal factors related to pregnancy, labor and delivery, or neonatal features such as race, gender, gestational age, birthweight, head circumference, and Apgar scores, were unrelated to the incidence of retinal hemorrhage. These results are in agreement with previous reports (2, 4, 5, 10, 12, 13, 18, 21, 25, 26).

The greater incidence of hemorrhage in babies born by vacuum-assisted delivery, and the frequent occurrence of unilateral hemorrhage, implicate a mechanical effect on retinal vessels during passage through the birth canal, perhaps by direct compression on the globe, in creation of retinal hemorrhage. However, if this were the sole mechanism, factors such as parity, episiotomy, head circumference, and

duration of labor might be expected to be associated with hemorrhage. Thus, maternal and fetal hemodynamic and rheologic changes during labor and delivery might also participate in the genesis of retinal hemorrhage, a suggestion consistent with hemorrhage in babies born via cesarean section, and with the occurrence of white-centered hemorrhages. These observations support the conclusion proposed by Planten et al that factors such as vacuum-assisted delivery may be a marker of difficult labor rather than the cause itself (27). Unfortunately, to date no adequate marker or combination of markers of RH have been noted to aid consistently in the retrospective determination of presence of neonatal RH. We submit that discrepancies between numerous studies that attempt to correlate several factors with RH reflect a multifactorial etiology for RH.

Approximately 90% of intra-retinal hemorrhage detected following birth resolved within two weeks, and none were detectable by four weeks after birth. One subretinal hemorrhage (which occurred in 1/298 eyes), persisted up to six weeks after birth. This study is the first to document, in a systematic fashion, the rate of disappearance of RH in newborns.

Birth-related RH has been considered in the differential diagnosis of RH associated with child abuse (43, 49). Both birth-related and traumatic RH may be present in all retinal zones. However, traumatic hemorrhage may occur in intraretinal, sub-retinal, and pre-retinal compartments (39, 44-46), whereas in our study no pre-retinal or vitreous blood was evident, and subretinal hemorrhage was rare and isolated. Other ocular signs of child abuse include periorbital ecchymoses or edema, conjunctival hemorrhage or abrasion, hyphema, nystagmus, and third nerve palsy (44), and are important because 21% of cases of child abuse with subdural hematoma do not involve RH (46). Our data indicate that intraretinal hemorrhage detected in infants older than one month of age is not likely related to birth, a finding that may be useful in the evaluation of suspected physical abuse or other ocular and systemic diseases. Therefore, the presence of RH in older infants should prompt the ophthalmologist to work with other members of the pediatrics team to investigate physical and social findings of child abuse.

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